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Abstracts

Morphogenesis

Program/Abstract # 112

Addressing the role of extrinsic cues in neuronal polarization

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We have previously described a novel role for the neuronal calmodulin binding protein GAP-43 as an important mechanistic component of the relationship between centrosome position and axon outgrowth (Mishra et al., Cell Cycle, 2008). However, the interaction between the polarizing cells and the extra-cellular cues governing polarity is not well defined. The position of GAP-43 in membrane micro-domains and its association with f-actin raises the possibility that GAP-43 might be one of the potential cell membrane proteins that interacts with extra-cellular cues that in turn leads to the establishment of neuronal polarity. Using micro-contact printing (μCP) technique, E-cadherin, Sonic Hedghog (Shh) and Vitronectin individually and in combination were printed onto coverslips. GAP-43 wild type (+/+) and knockout (−/−) enriched cerebellar granule cells were cultured on these coverslips and the response of the cells to the extra-cellular cues was studied in the presence and absence of GAP-43 by using a centrosomal marker as a readout of cell polarity. We show that the different printed proteins had differential effects on the orientation of the centrosome. In addition, in the absence of GAP-43 the effect of extra-cellular proteins on the orientation of the centrosome was abolished. These results show that GAP-43 is required to link centrosome position to extra-cellular molecular cues that is important for generating neuronal polarity in cerebellar granule cells.

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Program/Abstract # 113

Growth dynamics of clusterized neuronal network in vitro

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Time evolution of self-organized clusterized neuronal networks at high cell density in vitro is experimentally investigated using time-lapse phase contrast microscopy. Initially, homogeneously distributed cortical neuronal cells grow bundles to form a network in which each node is consisted of a single cell. Then the contractions of bundles make neighboring cells aggregate to form a hierarchical clusterized network of spherical clusters connected by giant bundles, in which each cluster is about a few tens of micro-meters in diameter and consisted of a few hundreds of neurons. In the morphogenesis after the initial formation of clusterized network, the cluster size and number can further evolve through the merging of some neighboring cluster pairs. The merging of two neighboring clusters causes the formation of the larger spherical cluster. The clusterized network can exhibit self-organized synchronous firing from the intra- to inter-cluster level. The detailed dynamical processes in the network formation are presented and discussed.

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Program/Abstract # 114

Twist1 is required for cardiac neural crest morphogenesis

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The basic helix–loop–helix (bHLH) transcription factor Twist1 regulates essential cellular functions in mesenchymal cell populations during embryonic development and in pathological disease. Development of the cardiac outflow track (OFT) into the functional aortic arch and pulmonary trunk is dependent upon the dynamic, coordinated contribution of multiple mesenchymal cell populations. Here, we report that *Twist1*$^{-/-}$ mice exhibit hypoplastic OFT cushions containing amorphic cellular nodules. This nodular mesenchyme is marked by expression of the related bHLH factors *Hand1* and *Hand2*, but not the normal cushion marker *Periostin*. Lineage mapping identifies nodule cells as exclusively of neural crest in origin, and further reveals a delay in neural crest OFT emigration. These mapping studies additionally uncover nodules in the pharyngeal arches and trace *Twist1*$^{-/-}$ neural crest cell-adhesive defects back to the dorsal neural tube, which exhibits an expanded domain of *Wnt1-Cre*-marked cells in the absence of overt defects in cell proliferation or cell death. Together, these data support a model where *Twist1* is required for proper cardiac neural crest cell morphogenesis and delamination from the dorsal neural tube. Within emigrating neural crest cell populations, a *Hand*-expressing subpopulation displays defective differentiation, tracking, and cell–cell interactions, further compromising cardiac neural crest cell colonization of the OFT.

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